Acute Myocardial Infarction

Urine Glutamic Oxalacetic Transaminase Activity

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THERE IS general agreement as to the diagnostic value of serum glutamic oxalacetic transaminase (SGO-T) determinations in patients with diseases of the heart, liver and skeletal muscle.* The ultimate fate of the serum enzyme in the body is unknown. Since blockage of the reticuloendothelial system with India ink does not result in increased serum activity, sequestration of this enzyme in those cells is apparently not significant.3 Hill and coworkers4 measured the excretion of this enzyme in the bile of rats following intravenous injection of transaminase. In the control animals, very small amounts of transaminase were excreted in the bile intermittently, the total amount never being more than 80 units in 10 hours; none of the rats excreted more than 1 per cent of the injected transaminase or apotransaminase. Therefore, in rats, the bile does not appear to play a significant part in the excretion of transaminase.4 Dunn and coworkers2 in experiments on normal mongrel dogs and dogs with artificially produced myocardial infarctions were unable to recover significant amounts of GO-T in the bile. The role of the kidney in the inactivation or excretion of this enzyme has been partially clarified. No measurable amounts of GO-T were detected in the urine of normal dogs after the intravenous administration of co-T or in dogs in which myocardial infarctions were produced; it was not stated whether these determinations were made on 24-hour urine specimens or on fresh specimens. The rate of disappearance from the serum remained approximately the same in dogs without kidneys, indicating a lack of inactivation by the kidneys. Rosalki12 found small amounts of lactic dehydrogenase (LD) and GO-T in the 24-hour urine specimens of normal persons and in three patients with acute myocardial infarction. However, in patients with acute renal disease, the urine LD to a moderate degree and the urine GO-T (UGO-T) to a lesser degree became elevated. There• The urinary content of glutamic oxalacetic transaminase (UGO-T) was determined in 16 consecutive patients with acute myocardial infarction. In all of them it was above normal.

In some patients the UGO-T remained elevated for a longer period than did the blood content of that enzyme.

It is possible that in certain patients with acute myocardial infarction the kidneys eliminate significant amounts of GO-T.

oxalacetic transaminase in freshly voided urine of normal persons and of patients with acute myocardial infarction because these determinations had not been previously reported. The enzyme activities were determined by the spectrophotometric method.⁷ The urine pH did not influence the activity of the enzyme in the urine. However, the activity of the enzyme slowly disappeared on standing, the decreased activity becoming apparent after 30 minutes of standing. Hence the determinations were performed within 30 minutes of collection. This reduction in activity was not constantly influenced by moderate refrigeration, but the specimens could be frozen for 48 hours without loss of any activity. The decreased activity on standing apparently accounts for the small amounts of this enzyme detected in 24-hour specimens in both controls and patients with acute myocardial infarction reported heretofore.

There was no apparent relationship between the presence of enzyme activity and the presence or amount of proteinuria. Urine determinations were made on 59 control patients with no evidence of acute disease of the heart, liver, or skeletal muscle. Only one determination was over 9 units; in 14 consecutive controls, the mean determination was 5.5 units per ml. of urine per minute with a standard deviation of 3.1. Sixteen consecutive patients with acute myocardial infarction were evaluated by daily determinations of the UCO-T. As may be seen in Chart 1, the UGO-T activity was elevated in all of these patients with acute myocardial infarction. The peak levels varied from 17 to 53 units. There did not appear to be a linear relationship between the

fore, we sought to evaluate the activity of glutamic

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^{*}References No. 1, 5, 6, 8, 9, 11, 13, 14.

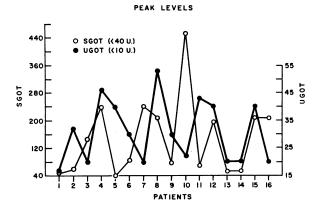


Chart 1 plots the peak levels of urine (heavy line) and serum (light line) oxalacetic transaminase activity seen in 16 consecutive patients with acute myocardial infarction.

height of elevation in the serum and in the urine. However, the charted curve of the content in the urine generally followed the curve of the serum content. The activity was usually elevated in the first 24 hours after infarction, reached a peak in 24 to 48 hours, and usually returned to normal in 72 to 96 hours. The ugo-T elevations were two to five times the upper limits of normal. Chart 2 depicts the duration of elevation of sgo-T and ugo-T. Although no clear-cut pattern is obvious, there is a tendency for the ugo-T to remain elevated for two to three days longer than the sgo-T.

REPORTS OF TWO CASES

The following case reports document many of the relationships mentioned above.

CASE 1. A 33-year-old white male physician had severe substernal chest pain at 1 a.m. An electrocardiogram was typical of a classical evolution of an acute transmural anteroseptal myocardial infarction. The daily sco-T and UGO-T determinations are shown in Chart 3. The UGO-T activity was elevated eight hours after the acute episode of chest pain and remained elevated for 20 days. There was no relation between the degree of elevation of UGO-T and sgo-T on any given day.

This case report demonstrates that the UGO-T may become elevated very early after a myocardial infarction, tend to follow the general course of the SGO-T, and remain elevated after the SGO-T has returned to normal.

CASE 2. A 44-year-old white man had acute myocardial infarction on June 25. As can be seen in Chart 4, the SGO-T remained elevated until June 30 while the UGO-T was still almost at the peak level on July 2, illustrating a tendency (noted in several patients) for the UGO-T and SGO-T curves to run parallel, but for the former to remain elevated longer.

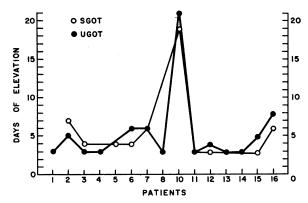


Chart 2 compares the duration of elevation of urine (heavy line) and serum (light line) glutamic oxalacetic transaminase activity in 16 patients with acute myocardial infarction.

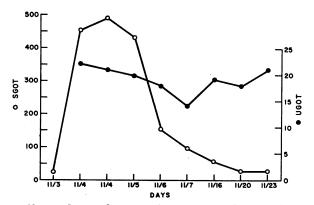


Chart 3 depicts the urine (heavy line) and serum (light line) glutamic oxalacetic transaminase activity in a patient with a severe myocardial infarction. Note that the UCO-T remained elevated for several days after the SCO-T returned to normal levels.

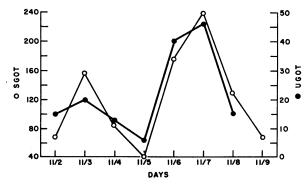


Chart 4 represents the pattern of the urine (heavy line) and serum (light line) glutamic oxalacetic transaminase activities in a patient with acute myocardial infarction. See text for discussion.

DISCUSSION

The diagnostic value of urinary GO-T determinations would appear to be obvious. The procedure is not only an additional method of diagnosing acute myocardial infarction without resort to multiple venipuncture, but in certain patients it may detect elevations after serum content of this enzyme has returned to normal.

It has been estimated that normal heart muscle contains 300,000 to 400,000 units of GO-T per gram of tissue and that 30 per cent may be lost during the first 24 hours after acute myocardial infarction; 10 since peak levels of 40 to 50 units per milliliter of GO-T in the urine were detected in a few of the patients in the present series, if we may postulate a urinary output of about 1,500 milliliters, these patients may have eliminated 70,000 units of GO-T via the kidneys. Thus, contrary to previous reports, the kidney may in some patients excrete a significant amount of this enzyme.

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REFERENCES

- 1. Agress, C., Jacobs, H. I., Glassner, H. F., Lederer, M. A., Clark, W. G., Wróblewski, F., Karmen, A., and LaDue, J. S.: Serum transaminase levels in experimental myocardial infarction, Circulation, 11:711, 1955.
- 2. Dunn, M., Martins, J., and Reissmann, K. R.: The disappearance rate of glutamic oxalacetic transaminase from the circulation and its distribution in the body's fluid compartments and secretions, J. Lab. Clin. Med., 51:259, 1958.
- 3. Henley, K. S., Schmidt, E., and Schmidt, F. W.: Serum enzymes, J.A.M.A., 174:977, 1960.

- 4. Hill, R. M., Nelson, B., and Sowl, D. D.: Excretion of glutamic oxaloacetic transaminase in the bile of rats, Fed. Proc., 19:108, 1960.
- 5. Kalmansohn, R. B., and Kalmansohn, R. W.: Acute nonspecific pericarditis: Reports of two cases with elevated serum glutamic oxalacetic transaminase activity, Calif. Med., 87:171, 1957.
- 6. Kalmansohn, R. B., and Kalmansohn, R. W.: An evaluation of serum glutamic oxalacetic transaminase activity in pericarditis, Am. Heart J., 55:739, 1955.
- 7. Karmen, A., Wróblewski, F., and LaDue, J. S.: Transaminase activity in human blood, J. Clin. Invest., 34:126, 1955.
- 8. Kattus, A. A., Jr., Watanabe, R., Semenson, C., Drell, W., and Agress, C.: Serum aminopherase (transaminase) in diagnosis of acute myocardial infarction, J.A.M.A., 160: 16, 1956.
- 9. LaDue, J. S., Wróblewski, F., and Karmen, A.: Serum glutamic oxalacetic transaminase activity in human acute transmural myocardial infarction, Science, 120:3117, 1954.
- 10. Lemley-Stone, J., Merrill, J. M., Grace, J. T., and Maneely, G. R.: Transaminase in experimental myocardial infarction, Am. J. Physiol., 183:555, 1955.
- 11. Ostrow, B. H., Steinberg, D., Tichtin, H. E., Polis, G. N., and Evans, J. M.: Serum glutamic oxalacetic transaminase in coronary artery disease: A review of 201 cases, Circulation, 14:290, 1956.
- 12. Rosalki, S. B., and Wilkinson, J. H.: Urinary lactic dehydrogenase in renal disease, Lancet, 2:327, 1959.
- 13. Sampson, J. J.: Serum transaminases and other enzymes in acute myocardial infarction, Prog. Cardiovasc. Dis., 1:187, 1958.
- 14. Wróblewski, F.: The clinical significance of transaminase activities of serum, Am. J. Med., 27:911, 1959.

